Modern Concepts of Cardiovascular Disease

Published monthly by the AMERICAN HEART ASSOCIATION

1775 BROADWAY AT 58TH STREET, NEW YORK, N. Y.

DR. THOMAS J. DRY, Rochester, *Editor*

DR. HOWARD B. BURCHELL, Rochester, *Associate Editor*

VOL. XVII **MAY, 1948** **No. 5**

DISTURBANCES OF WATER AND SODIUM BALANCE IN CONGESTIVE HEART FAILURE

As indicated by Wheeler and White (Modern Concepts of Cardiovascular Disease, Vol. 16, No. 10, October, 1947), studies of the disturbances in water and sodium balance in congestive heart failure have resulted in the restriction of sodium in therapy. They also have shown the relative unimportance of restriction of fluid; though forcing of water may cause the patient discomfort, it is not harmful, provided that the intake of sodium remains low. These conclusions have been reached mainly through clinical experience and from limited experimental and theoretic data. Still unknown are many important factors operating when sodium is limited, and especially when measures which increase the rate of excretion of sodium, such as administration of ammonium chloride and mercurial diuretics, are employed. Criteria which should govern the administration of these therapeutic procedures have not been outlined, largely because of lack of understanding of the mechanisms concerned in the production of congestive heart failure. Some of the problems involved in the mechanism of congestive heart failure will be outlined briefly.

Although many important aspects of congestive heart failure remain unknown, most physiologists and clinicians support either the "forward" or "backward" failure concepts or a combination of these as the fundamental mechanism of congestive heart failure. Considerable data have been accumulated over many years which cast doubt on the adequacy of the classical concepts for explaining the mechanism of congestive heart failure. Though many of these data may not diverge from the classical concepts, our present state of knowledge fails to reveal sufficient relationships. Many observations which seem to be entirely unrelated are worthy of mention herein.

Before presentation of these observations, which are difficult to interpret in the light of the classical concepts of the mechanism of congestive heart failure, it is necessary to review each concept briefly. The forward failure theory is based primarily on the concept that the cardiac output is decreased; it is, therefore, often referred to as the "decreased cardiac output theory." The diminished cardiac output produces anoxia in tissues, which in turn effects increased capillary permeability and edema, with the classical clinical picture of congestive heart failure. Therefore, this theory requires the following conditions: 1. The cardiac output must be reduced during the period of failure, certainly during intervals of physical activity. 2. The cardiac output must increase with compensation. 3. States which reduce cardiac output must increase the severity of the failure. These postulates have not been definitely established.

The backward failure theory states that the affected ventricles fail to pump the blood which reaches them, so that it accumulates in the ventricles, in the atrium immediately behind and in the veins emptying into that particular atrium. Increased hydrostatic pressure within the capillaries, circulatory stasis, capillary anoxia, increased capillary permeability and the syndrome of congestive heart failure with edema ensue. This mechanism would require at least: (1) reduction in cardiac output at some time, (2) relatively high protein concentration in the edema fluid (normal approximately 0.5 gm. per 100 c.c.) and (3) increase in venous pressure before the onset of congestive heart failure.

Many observers believe that both theories are required to explain the syndrome of congestive heart failure.

Among observations which are difficult to explain by means of the classical theories are the following:

1. Ligation of the inferior vena cava often results in elevation of the venous pressure in the legs to more than 600 mm. of mercury without development of clinical evidence of edema.

2. Though intracapillary pressure in the feet of a man standing is greater than that in the arteries of the arms, clinical evidence of edema in the feet is usually absent or mild even after prolonged periods in the upright position.

3. The intense anoxia of the capillary endothelium associated with tetralogy of Fallot, high altitudes, and chronic pulmonary disease does not necessarily result in the syndrome of congestive heart failure.

4. The clinical state of the syndrome of congestive heart failure may vary independent of demonstrable changes in venous pressure.

5. The exact correlation of the state of the syndrome of congestive heart failure with cardiac output has not been established.

ANNUAL DINNER

The Annual Dinner of the American Heart Association will take place in the Grand Ballroom of the Palmer House, Chicago, Illinois, 7:30 p.m., Saturday, June 19, 1948. Unless otherwise requested, all tickets for reservations at $7.00 each will be held until the day of the Dinner.

6. The protein concentration of the edema fluid in congestive heart failure is not elevated, a fact which is incompatible with increased capillary permeability.

7. There is no correlation between the plasma proteins and the state of congestive heart failure.

8. Extensive destruction of cardiac muscle in experimental animals by burning and ligation of the coronary arteries does not result in the syndrome of congestive heart failure.

9. A mercurial diuretic or a low salt diet or both, which have extra-cardiac action, usually effect remission in the syndrome of congestive heart failure.

10. An overdose of desoxycorticosterone acetate results in a clinical state which resembles the syndrome of congestive heart failure, but is not associated with any known direct cardiac effects.

Data which suggest other mechanisms concerned with the development of congestive heart failure also have been reported. It has been definitely established that the *renal function* is altered in congestive heart failure. As early as 1722 Baynard suggested renal influence. Abnormal urinary findings, that is, casts, hematuria, albuminuria and reduced renal function, are known to be associated with congestive heart failure. Oliguria with increased concentration of the urine occurs during failure, whereas diuresis is associated with compensation. The effect of mercurial and other diuretics on congestive heart failure are extracardiac, their action being mainly renal and possibly extrarenal and generalized. Restriction of sodium results in improvement in cases of cardiac failure, whereas its administration causes exacerbation of the syndrome. Studies have indicated a diminution in renal blood flow during failure, followed by altered renal function and reduction in the rate of excretion.

Experiments concerning the rate of excretion of sodium conducted by means of radiosodium (Na^{24} and Na^{22}) have shown definite retention of the element during failure; this retention is most marked when failure is developing or becoming progressively worse. Periods of compensation are accompanied by sodium diuresis. Administration of sodium does not result in increased excretion of sodium; frequently greater retention of sodium ensues. Tracer studies have shown that the normal person excretes half of the total body sodium in about fifteen days, whereas the patient with chronic congestive heart failure does it in about forty days. The turnover of sodium varies with the phase of the failure; it is most rapid during compensation and least rapid during exacerbation. Other factors are also concerned with the nature of the turnover. Patients with the nephrotic syndrome of chronic glomerulonephritis also show a reduced rate of excretion of sodium. The turnover is related to the degree of renal disease. These patients may require more than sixty days to excrete half the body sodium. The rate of excretion or turnover of sodium in the patient who has congestive heart failure can be brought to normal levels by means of mercurial diuretic agents which have no direct cardiac action. In the normal subject, it may be reduced to levels found in congestive heart failure by means of restriction of intake of sodium or by administration of desoxycorticosterone acetate; neither of these has any direct cardiac action. Many problems related to electrolyte and water metabolism in congestive heart failure are still unsolved and require elucidation for a clear understanding of this condition.

The mechanism of congestive heart failure is still controversial, though many important physiologic phenomena have been described. The two classical theories are of considerable significance as tools for thought but do not adequately explain the physiologic disturbances. Much more intensive investigation is necessary for a better understanding of the mechanism of failure, thereby leading to proper management and prevention of the syndrome. Observations indicate that the factors concerned with failure are most likely to be complex and to be related to chemical agents, such as hormones and enzymes, which are generalized in their action and which may have direct or indirect influence on renal function so as to increase the degree of tubular reabsorption of water and sodium as well as other substances from the glomerular filtrate. The trigger mechanisms which initiate the complex diffuse physical and chemical phenomena probably differ from patient to patient and most likely include such factors as increased venous pressure, decreased cardiac output and anoxia among many other possibilities.

That sodium diuresis and restriction of intake of sodium benefit the patient with congestive heart failure is generally accepted. There are, however, many important aspects of these forms of therapy which need clarification in order that their use may not result in serious damage to patients. Clinical criteria should guide the degree of sodium restriction and the administration of diuretics. There is no doubt that requirements vary with individual patients and within the same patient. Considerable harm may follow injudicious therapeutic measures; selection of therapy for individual patients must be placed on a more satisfactory plane. It is not known to what extent restriction of sodium and diuresis prolong life, although certainly they do promote symptomatic improvement. Additional investigation of congestive heart failure will elucidate these and other challenging problems in this disease.

George E. Burch, M.D.
New Orleans, Louisiana

PROGRAM
of the
TWENTY FIRST SCIENTIFIC SESSIONS
of the
AMERICAN HEART ASSOCIATION
June 18-19, 1948

North Ballroom, Stevens Hotel, Chicago, Illinois

FIRST SESSION
2:00 P.M., Friday, June 18

Chairman: Arlie R. Barnes, M.D., President, American Heart Association
Secretary: Harry E. Ungerleider, M.D.

1. A CLINICAL STUDY OF SUBACUTE BACTERIAL INFECTION CONFINED TO THE RIGHT SIDE OF THE HEART OR THE PULMONARY ARTERY.
   Paul S. Barker, M.D., Ann Arbor, Michigan.
2. PLASMA AND BLOOD INFUSION FOLLOWING MYOCARDIAL INFARCTION.
   John J. Sampson, M.D. and Isidore M. Singer, M.D.. San Francisco, California.
3. REPORT OF THE COMMITTEE FOR THE EVALUATION OF THE USE OF ANTICOAGULANTS IN THE TREATMENT OF CORONARY THROMBOSIS WITH MYOCARDIAL INFARCTION.
   Irving S. Wright, M.D., Chairman, New York, New York.
4. ON THE OPTIMAL INTAKE OF WATER FOR EDEMATOUS CARDIAC PATIENTS.
   Ferdinand R. Schemm, M.D., Great Falls, Montana.
5. SURGICAL TREATMENT OF ANEURYSMS.
   Gerald H. Pratt, M.D., New York, New York.
6. THE DIAGNOSIS OF INTERAURICULAR SEPTAL DEFECT.
   Richard S. Cosby, M.D. and George C. Griffith, M.D., Pasadena, California.
7. STUDIES OF ARTERIAL OXYGEN SATURATION IN PATIENTS WITH SUSPECTED ARTERIAL HYPOXEMIA WITH USE OF A MODIFIED OXIMETER.
   J. E. Geraci, M.D., George E. Montgomery, Jr., M.D., Thomas Dry, M.D., and Earl H. Wood, M.D., Rochester, Minnesota.

5:00 P.M., Friday, June 18

Annual Business Meeting:

a) Election of Officers and Assembly.

b) Approval of changes in Certificate of Incorporation.

SECOND SESSION
9:00 A. M., Saturday, June 19

Chairman: M. C. Winternitz, M.D., Chairman, Section on Peripheral Circulation
Secretary: Grace M. Roth, Ph.D.

8. ARTERECTOMY IN THE TREATMENT OF ISCHEMIC NEURITIS FOLLOWING ACUTE ARTERIAL OCCLUSION.
   Norman E. Freeman, M.D., Richard E. Gardiner, M.D., and Frank H. Leeds, M.D., San Francisco, California.
9. GEORGE BROWN MEMORIAL LECTURE: ON THE NATURE AND TREATMENT OF SHOCK.
   Irvine H. Page, M.D., Research Division of the Cleveland Clinic Foundation, Cleveland, Ohio.

I enclose $.....for.....reservations at $7.00 each for the Annual Dinner of the Association, to be held at the Palmer House, Chicago, Illinois, on Saturday, June 19, 1948.

Name.....

Street and Number.....

City and State.....

(Please make checks payable to American Heart Association, Inc.)

10. DIFFERENTIATION OF THE CHANGES IN THE Q-T INTERVAL IN HYPOCALCEMIA AND HYPOPOTASSEMIA.
A. Carlton Ernstene, M.D. and William L. Proudfit, M.D., Cleveland, Ohio.

11. RADIOCARDIOGRAPHY: A GRAPHIC METHOD FOR STUDYING THE FLOW OF BLOOD THROUGH THE HEART BY MEANS OF RADIOACTIVE ISOTOPES.
Myron Prinzmetal, M.D., Eliot Corday, M.D., Ramon Spritzler, M.D., and H. C. Bergman, Ph.D., Los Angeles, California.

12. THE WATER TOLERANCE OF THE HYPERTENSIVE PATIENT: ITS RELATION TO OPERABILITY.
Geza de Takats, M.D. and Edson F. Fowler, M.D., Chicago, Illinois.

12:00—1:00 P.M., Saturday, June 19

Panel Discussions:

a) THE ANTICOAGULANT TREATMENT OF CORONARY ARTERY DISEASE AND ANTICOAGULANT THERAPY IN GENERAL. North Ballroom
Irving S. Wright, M.D., Chairman

b) CRITICAL EVALUATION OF NEW VASODILATOR DRUGS. Room #2
N. C. Gilbert, M.D., Chairman

If you wish to attend a panel discussion, you are invited to forward a question or topic which you would like to have discussed to the American Heart Association, 1775 Broadway, New York, New York.

THIRD SESSION

2:00 P.M., Saturday, June 19

Chairman: Herrman E. Blumgart, M.D., Chairman, Program Committee
Secretary: Harry E. Ungerleider, M.D.

13. CLINICAL STUDIES ON TWENTY CASES OF COARCTATION OF THE AORTA.
M. J. Shapiro, M.D., Minneapolis, Minnesota.

14. NORMAL AND IMPAIRED FUNCTION OF THE SUPERFICIAL LEG VEINS.
J. B. Hickam, M.D. and R. P. McCulloch, M.D., Durham, North Carolina.

15. PATHOLOGY OF THE PULMONARY ARTERIOLES IN AORTIC COARCTATION ASSOCIATED WITH PATENT DUCTUS ARTERIOSUS.
Jesse E. Edwards, M.D., John A. Douglas, M.D., Howard B. Burchell, M.D., and Norman A. Christensen, M.D., Rochester, Minnesota.

16. PRODUCTION OF ARTERIOSCLEROSIS IN DOGS BY CHOLESTEROL AND THIOURACIL FEEDING.
Alfred Steiner, M.D., Forrest E. Kendall, Ph.D., and Margaret Bevans, M.D., New York, New York.

17. BIOMICROSCOPY OF CONJUNCTIVAL VESSELS IN HYPERTENSION.
Arthur Lack, M.D., Travis Winsor, M.D., William Adolph, M.D., and Walter Ralston, M.D., Van Nuys, California.

18. CORRELATION BETWEEN THE DECREASE IN CORONARY FLOW AND THE APPEARANCE OF ELECTROCARDIOGRAPHIC CHANGES.
Rene Wegria, M.D., Marcel Seegers, M.D., and Richard P. Keating, M.D., New York, New York.

7:30 P.M., Saturday, June 19

Annual Dinner
Grand Ballroom, Palmer House, Chicago, Illinois
Speaker: Mr. Thomas I. Parkinson, Chairman, Board of Directors;
President, Equitable Life Assurance Society of the United States.

PROGRAM COMMITTEE

Chairman: Herrman E. Blumgart, M.D., Boston

Emmet B. Bay, M.D., Chicago
George E. Burch, M.D., New Orleans
Douglas Deeds, M.D., Denver
Francis R. Dieuaide, M.D., New York
A. Carlton Ernstene, M.D., Cleveland
Arild E. Hansen, M.D., Galveston
William P. Holbrook, M.D., Tucson
Franklin W. Johnston, M.D., Ann Arbor
William B. Kountz, M.D., St. Louis
Edwin P. Maynard, Jr., M.D., New York
Teresa McGovern, M.D., New York
Johnson McGuire, M.D., Cincinnati
Rustin McIntosh, M.D., New York
Franklin R. Nuzum, M.D., Santa Barbara
Myron Prinzmetal, M.D., Beverly Hills
John J. Sampson, M.D., San Francisco
Harold N. Segall, M.D., Montreal, Canada
Helen Taussig, M.D., Baltimore
Francis C. Wood, M.D., Philadelphia
Wallace M. Yater, M.D., Washington, D.C.

NOTES

NOTES